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EARLY DETERMINATION OF GALL BLADDER DISEASE AND ITS TREATMENT*

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It has been estimated that about sixty per cent of people who consult an M. D. do so because of indigestion, and that twenty per cent of cases with indigestion as the chief complaint have gall bladder disease.

It occurs three times as frequently among women as among men and the most commonly accepted as predisposing factors are typhoid fever and pregnancies.

When such figures as 75% of women operated upon for gall bladder disease are women who have borne children are quoted, it seems that the same could be said of all adult diseases, but certainly the stasis and cholesterol disturbance during pregnancy probably do play a part. As to typhoid fever, the connection has been proven many times by the recovery of typhoid bacilli from gall stones, but I believe that many of our infectious diseases are also irritating factors in the pathology.

As to age incidence, we frequently tend to place it after forty. This was partly because of the saying attributed to the late Dr. John B. Deaver "Fair, fat and forty, and belches gas"—was supposed to be pathognomonic of gall stones. It is true that most cases do manifest trouble after forty, yet gall stones have been reported as early as seven years of age. In our experince we have seen them under twenty years of age, and quite young cases of troublesome digestive symptoms where we felt the gall bladder was at fault. Today, the "bilious child" formerly treated with calomel and now given milk of magnesia is probably the forerunner of our adult case with numerous small stones.

As to race, a curious about-face has occurred in reference to gall bladder disease in the colored race. As students we were taught that gall bladder disease did not occur among the colored. We were not long out of school before we found this teaching quite falacious. If the figures are taken in proportion to the ratio of population, the figures are only slightly less in the colored race.

The criteria for diagnosis of cholecystic disease for which operation is required include:

1. A satisfactory account of one or more attacks of biliary colic or its equivalent, with or without fever, chills or jaundice.
2. Residual tenderness in the region of the gall bladder following such painful episodes.
3. Flatulent indigestion with distress after full meals, especially of fatty or coarse foods.
4. Cholecystographic evidence of a nonfunctioning gall bladder or the presence of stones.
5. Reasonably exact exclusion of conditions that stimulate cholecystitis.

The classical symptom of cholecystitis with or without stones, is, as we all know, biliary colic. This is characterized by an unusually severe pain, originating in the upper right quadrant of the abdomen and extending to the right subscapular region. On occasion, however, it may occur in the epigastrium, the upper left quadrant, or it may originate in the back and simulate lumbago. Belching, nausea, and vomiting usually accompany the pain, along with chills, fever, and leukocytosis. Icterus following an attack is particularly significant of the presence of stones. Also the residual tenderness, soreness and slight fever are indicative of a genuine gall bladder attack. The modes of obtaining relief are worth mentioning, as morphine is usually the only drug capable of relieving the pain in an attack of colic of any severity.

The dyspepsia associated with cholecystic disease is usually described as gaseous indigestion, with distension and belching particu-

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larly noticeable after a full meal, especially when fats and roughage are eaten.

CLASSIFICATION OF CHOLECYSTIC DISEASE

The commonest and most severe forms of cholecystitis are those which are associated with stones, stones being present in the gall bladder in from 90-95% of all acute cases. The symptoms of acute cholecystitis with stones are as a rule much more severe than in the non-calculus form, and may come on much more suddenly. This is true even in the absence of few or all symptoms of previous cholecystic disease. It is here that the attack is likely to be much more typical, with severe upper right quadrant pain and tenderness, muscular rigidity, vomiting, abdominal distension, chills and fever.

Other less common forms of cholecystic disease are:

1. Acute catarrhal cholecystitis. The clinical picture of this type is quite variable. The condition may be very mild and pass unrecognized. If recognized clinically, the onset is usually gradual, with some nausea, chills, slight fever, along with gradual increase of upper right quadrant pain and tenderness. The attacks usually subside with resolution and recovery. Jaundice may or may not be present, depending on the extent of involvement of the biliary tract.

2. Acute infectious cholecystitis, probably hematogenous in origin, is a rather rare condition, but much more severe in symptoms than to the catarrhal type. The incidence of perforation is much higher in this type, and carries a much higher mortality.

3. Acute septicemic cholecystitis, a rare condition occasionally associated with subacute bacterial endocarditis, osteomyelitis, etc., in which the clinical picture is that of an acute fulminating cholecystitis with the wall of the gall bladder containing definite areas of infarction.

4. Acute typhoidal cholecystitis, occurring during the course of typhoid, is generally characterized by superimposing the symptoms of a gall bladder attack on top of the picture of typhoid. Perforation and suppuration are particularly likely to occur in these cases. A similar condition has also been described in connection with pneumonia, cholera, scarlet fever, erysipelas, and variola.

5. Puerperal forms of cholecystitis are fairly common, and may be catarrhal, suppurative, hydrops, or even gangrene and perforation. This condition, is usually one superimposed on pre-existing stones in the gall bladder.

6. Gangrenous cholecystitis is a condition usually associated with a blocking of the cystic duct by stone, with a superimposed highly virulent organism.

7. Chronic non-calculous cholecystitis. A form which produces a great variety of less severe symptoms and which is much more difficult to describe and diagnose. Cholecystograph usually shows some positive findings but nothing as conclusive as in the other forms.

The stasis gall bladder belongs in this group and is defined as an anatomically normal organ which physiologically so functions that it produces dyspepsia, colic, and other symptoms of cholecystic disease.

8. T. B., syphilis, actinomycosis, parasitic infection of the gall bladder—all of these are very rare.

9. Tumors of the gall bladder.

(a) Papillomas—present in 8-10% of all surgically removed gall bladders, usually small, and not of much importance.

(b) Primary carcinoma. Generally associated with stones, but occurs in less than 1% of all cases of diseased gall bladders. The clinical picture is extremely variable, usually not diagnosed except at operation or autopsy. The prognosis is very poor, the patient usually dying within a year, even with early surgical removal.

In making the differential diagnosis of gall bladder disease, the history is of much more importance than any physical findings. Of course, if an x-ray study has been made, or an abdominal operation has been done and gall stones found, it is more positive than history.

In the history, the character, situation and severity of acute attacks of pain, the sequelae of any such attacks, and the complaint of flatulent dyspepsia are most suggestive. Though patients frequently deny previous gall bladder attacks, they readily remember previous bouts of acute indigestion or gastric neuralgia, so diagnosed by the then attending physician. Tenderness, protective rigidity,

and fever are early symptoms. Violent vomiting, severe pain directed across the upper abdomen, dark urine, light stools, and puritis identify the attacks as involving the common bile duct before true clinical jaundice may be noted.

In the acute stage of gall bladder disease, and in the presence of increasing jaundice, the cholecystographic study should not be requested. When the dye study is done, we have never been satisfied with either single or double oral dose in the final diagnosis of non-functioning gall bladder. Repeating with intravenous dye too frequently reveals a well functioning organ.

A report of poorly functioning or non-functioning gall bladder, or the presence of shadow indicating stone or stones is almost positive proof of cholecystic disease. On the other hand, a negative report does not rule out disease. We all know that not all gall stones are opaque to x-ray, and that a gall bladder may still give a good functioning x-ray result yet be diseased to the point of giving rise to biliary colic.

During the last decade cholecystography has become an exceedingly reliable aid in the diagnosis of cholecystic disease. Statistics compiled at the Mayo Clinic show that from 90-95% of cholecystographic diagnoses, whether positive or negative, are correct. Of these patients whose cholecystograms are abnormal, more than 98% have disease of the gall bladder. Of those patients who have gall stones, about 99% yield positive cholecystographic data. On the negative side, however, about 10% of those cases with a normal cholecystogram have a diseased gall bladder.

As our clinical experience increases, we realize that laboratory findings that support our clinical diagnosis are a satisfaction, but if they do not support such diagnosis it does not necesarily mean that the clinical diagnosis is wrong.

Acute gall bladder disease with its pain, nausea and vomiting must be differentiated from renal colic. Not too infrequently a flat plate will show the presence of both types of stones. Pancreatic calculi have confused the picture, but not as frequently as kidney stone.

Any thing giving rise to acute pylorospasm such as ulcer and polyp must be considered.

In cases of long standing symptoms, the x-ray differential should include gastric ulcer, duodenal ulcer, gastric polyp, gastric carcinoma, and the possibility of pyelonephritis must be considered. Recently a case was reported that only on third admission to a hospital was the trouble found to be kidney instead of gall bladder. Two negative cholecystograms and exploratory operation were done before the true diagnosis was made.

Concerning the differential, between gall bladder and carcinoma, it is rather appalling to analyze the number of cases previously operated upon where the real offender has been an adjacent carcinoma. In carcinoma of the cecum, most patients have had a recent appendectomy; in carcinoma of the splenic flexure, a previous operation on the stomach is recorded; in carcinoma of the sigmoid and rectum, most of them have had a hemorrhoid operation within a year, while carcinoma of the hepatic flexure cases frequently show comparatively recent cholecystectomy.

Acute cholecystitis is frequently diagnosed in such cases as hiatal hernias, acute mediastinitis, esophageal rupture and, spontaneous pneumothorax.

Coronary masquerades, where anginal attacks are frequently ascribed to acute indigestion or an acute gall bladder attack, are frequent enough for all of us to be on the alert.

Parenchymal diseases of the liver, abdominal crises of tabes dorsalis, and lead poisoning must likewise be eliminated by history, physical examination, or laboratory studies.

In other cases hemolytic icterus, gastro-intestinal allergy, and lesions of the appendix and cecum may prove quite confusing.

If one were satisfied to make a simple diagnosis of acute surgical abdomen and do the differential under palpation and direct vision, time might be saved in some few instances, but we are certain the incidence of error would certainly be greater.

The greatest percentage of operative failures in all types of surgery occurs where the operation is performed for relief of pain rather than the correction of proven disturbances in physiology. The longer the abdominal pain lasts the more likely the operative procedure may be a failure, for in over fifty per cent of cases where the pain lasts over

three months the pain is in the abdominal wall and not in the intra-abdominal viscera.

We still have the argument as to the value of early surgical intervention in gall bladder disease. The advocates of early intervention are correct, provided they can operate within the first six to twenty-four hours of an attack. If later than that the amount of edema complicates exposure, and surgical procedures lead to incomplete measures and secondary operations.

Early diagnosis of disease of the gall bladder is most desirable as shown by mortality and morbidity tables. It has been shown that gall bladder operations performed after one or two attacks carry a mortality of 3. to 3.5%; after 4 or 5 attacks, 6.7%; while those with jaundice have a 10 to 11% mortality.

The incidence of perforation of the gall bladder, acute suppurative cholecystitis, empyema of the gall bladder, and common duct obstruction are all much higher in patients with long standing history of cholecystic disease and repeated attacks of biliary colic.

Several years ago it was reported that the mortality rate in gall bladder disease was not proportionate to age, physical condition, number of attacks, or any one of many factors, but was directly proportionate to the number of hours which had elapsed from the onset of the acute attack until hospitalization. The big difference that occurs between home and hospital treatment, is the ability to control intake in the hospital much better than at home.

If the patient arrives 6 to 24 hours after beginning of an attack consideration may be given to immediate operation. If the diagnosis has yet to be proven or if more time has elapsed it is preferable to have the acute attack subside before considering surgery.

The first step in the treatment is to put an absolute stop to everything by mouth. This means that intravenous therapy should be almost continuous, consisting of glucose, some form of Vitamin K, and preferably mixed vitamins. Only in cases of severe wasting do we add amino acids.

Sedation and possibly heat over the gall bladder may add to patient's comfort. If the temperature shows a tendency to fall and tenderness subside, in three or four days the patient is usually ready for a check up by x-ray and reexamination of blood chemistry.

If the temperature does not subside promptly, or increases with increased pain and tenderness, one must consider the possibility of perforation of the gall bladder, with its resulting peritonitis and increased threat to life. Perforation is reputed to occur in 15% of cases, but fortunately the omentum prepares to plug the perforation by surrounding the diseased gall bladder even before perforation occurs. The difference in these two situations of course dictates a difference in the surgical treatment.

In the subsiding case, a time can be elected for exposure and deliberate surgery, usually the removal of the gall bladder, exploration of the common duct, peritonealization of the gall bladder bed and exploration of the abdomen, with possible appendectomy.

In the type which progresses in spite of putting the pylorus at absolute rest, the surgery becomes an emergency procedure, calling for drainage of the gall bladder, removal of the cystic duct obstruction or common duct obstruction, drainage, and out. The sequence of events after this type of surgery varies greatly, from absolute cure to the patient needing one or more operations before normalcy is reestablished. The commonest complication is persistence of biliary fistula which invariably means that there is still a stone in the ampulla or in the cystic duct. Of course this refers to those cases without jaundice.

This complication is caused by the edema present at the time of drainage being sufficient to prevent the recognition of the stone. It is that same edema which necessitates drainage rather than removal.

There is one curious complication of gall bladder disease that must be borne in mind, even though it is comparatively rare. This is intestinal obstruction due to gall stone. This occurs at any age, from 31 to 76 in the ten cases reported from Mayo Clinic. In their cases five had no symptoms prior to the onset of ileus. Three had a characteristic gall bladder history, two had only vague complaints, five had suffered from attacks of partial obstruction prior to onset of actual ileus. In each case the stone had migrated through a

cholecystenteric fistula—nine into the duodenum, one into the hepatic flexure. In none of the cases was their diagnosis made before operation. The stone was found in seven cases at operation, and in three cases the obstructing lesion was not found until autopsy was performed.

In contrast to the enormous amount of clinical work done in the Mayo Clinic, our own experience has been unusual, for we have had five such cases. In three cases the diagnosis was made prior to operation, two of these being definitely proven preoperatively by x-ray. The stone was removed in all five cases, but death resulted in two. In one fatal case the stone had lodged in the terminal ileum, about 12 cm. from the ileocecal valve, and the intervening bowel was gangrenous. In the other fatal case the stone was near the junction of the jejunum and ileum. Five days had elapsed from the time of onset of symptoms and patient died within eight hours of operation of an acute pulmonary edema. In four of these five cases the stone was in the last five inches of the ileum. In none of our cases of intestinal obstruction that have been autopsied has a gall stone been found at autopsy, as far as we know.

The usual procedure here is to move the stone back from its lodged position, apply clamps above and below, remove the stone and drain the distended bowel through a catheter brought out through a stab wound, well away from the operative incision.

We have limited our remarks to the presentation to the early diagnosis of gall bladder disease and have not attempted to discuss the diagnosis of early gall bladder disease. This is the realm of the internist and the gastroenterologist. As surgeons we are interested only in presenting the picture as the general practitioner sees it, and when he must decide between medical management and surgery.

2029 *Pine Street.*

DISCUSSION

DR. WILLIAM SPEER (Wilmington): What has been your experience, doctor, with acute pancreatitis following a long continued gall bladder history?

DR. BATES: Either because I do not recognize acute pancreatitis or because it does not happen to be sent into our hospital, I have seen, I think, less acute pancreatitis than anybody doing the same amount of work that I happen to be doing—in fact only two cases. Both cases were of real, severe acute pancreatitis, amounting almost to gangrene. We put in multiple drainage with scarifications. Both cases provided a long neglected gall bladder history, and I think there is a very definite relationship, because it is secondary to blockage of the pancreatic duct from the original pancreatic disease, but I have only the two cases to back my assertion.

DR. SPEER: The reason I asked is that within the last six weeks I was called to see a patient whom I had seen in definite gall bladder attacks for the last 15 years. She always refused hospitalization; she refused everything, because as soon as the pain was gone she was well. About six weeks ago, she was taken one night with another attack of what she thought was just her chronic gall bladder pain. They did not bother me at night; but called me in the morning. I could not get there at once, and when I finally got there one of the kids in the neighborhood said that the lady had been taken to the hospital. I went over to the hospital and the intern had made the same diagnosis—acute gall bladder—and given her morphine. She was calm and collected. I said to the husband: "Here is where we are going to keep her and study her, and get rid of her gall bladder condition," to which he agreed, and to which she agreed. I saw her that night, again. The nurse said she was comfortable and asleep; that it was not necessary to disturb her. I didn't. Next day I called in the morning; they said she was just about the same. I told them to take a flat plate of the abdomen, and in the afternoon I went to the x-ray laboratory, Dr. Lattomus said: "Well, I don't know as we can give you much help, as there is no visible evidence of calculi." While she was in the x-ray department she had an apparent typical gall bladder spasm. When I saw her I found she was in shock, and instituted treatment for a shock. Dr. Edgar Miller saw her with me at eight o'clock. She was still in shock, so we treated her for a couple of hours, trying to get her in better shape, with no result. At ten o'clock I operated. I never had thought of acute pancreatitis, because I had

had the picture of gall bladder for fifteen years. Dr. Miller thought perhaps she might have a ruptured ulcer, but when scrubbing up I said: "I can't see an ulcer picture or a gall bladder picture." It never occurred to me the woman might have an acute pancreatitits, until that time. The moment I opened the abdomen I got the characteristic fluid, and the diagnosis was evident. She had a pancreatitis, undoubtedly, which was the result of a long continued gall bladder disease.

It is the only one that I have seen for several years. Dr. Jones said that he had one the other night. I think there is no question but that this case was a direct result of a chronic gall bladder condition.

DR. HOWARD S. RIGGIN (Seaford): There are sections in the United States known as the "goitre belt." Well, I think Sussex county is the "gall bladder belt."

I had the Tuberculosis Pavilion years ago, and in those days forced feeding and rest was about the only thing we knew about Tb. That meant six or eight or ten raw eggs, in milk, a day. Well, you never saw so many bilious people in your life, and we had to stop all that.

Whether or not the people down in Sussex have classified me as a "gall bladder fanatic" I don't know, but so many of the women patients in the county get the same treatment and were told the same thing that maybe I do get more of the gall bladder cases. But, really, I have seen more gall bladder cases in Sussex county in the last six years than I saw in my last fifteen years in New York City.

But what I wanted straightened out is—I have a diet I put them on. As to things that hurt these cases, I have listed eggs, coffee, fat, of course, chocolate, raw onions, raw apples and salt fish. This, of course, is only from my own observation.

And another thing I checked on, not for many years, but maybe fifteen years—I noticed numerous gall bladder conditions developing in comparatively young people who have had their tonsils out. Now, has that anything to do with the infection that might go down and infect the gall bladder, or is it just my imagination? And is the fact that I have ruled against eggs—I have taken everybody off eggs that had any indigestion—is that just my imagination? I firmly believe people can handle roast beef better than eggs, yet the first thing they give you in convalescence is fresh eggs. Has this diet any significance or is it just merely my imagination?

As to the tonsils, I can't say that I have checked that long enough, but I have checked it enough to know that I have had cases where in six or eight years following a tonsilectomy, they definitely had a gall bladder condition.

DR. W. EDWIN BIRD (Wilmington): As to the diagnosis of pancreatic disease, and especially as to the differention between that and a gall bladder condition, I learned in my internship days that acute pancreatitis gives, frequently, a girdle sensation. I have seen this borne out several times, clinically. If you can rule out spinal cord disease this girdle sensation is almost pathognomonic of the pancreas. If a woman complains of a sensation as though there were a corset string around her waist, examine her neurologically to rule out disease of the spinal cord, and if you can do that, it's a pretty good bet you are dealing with a pancreatitis, or with a pancreatic cyst.

DR. BATES: Speaking of the "belt"—the thyroid or gall bladder belt—I do not know that there is anything in the bacteriology of a certain belt giving rise to conditions such as the water giving rise to thyroid disease: I think it is more the dietary habits of the district or the person. That is borne out by the fact that most of the largest clinics started as thyroid clinics. When they started the Lahey Clinic in Boston people felt it couldn't help but be a failure there, because people in Boston didn't have thyroid disease, but they have made out pretty well there. And in a district where eggs are a prominent part of the diet, and the poultry and meat have been denied them on account of the war conditions, I think there is no doubt that there has been an increase in gall bladder disease, or of precipitating gall bladder disease. I agree with what the doctor said about meat eating, if, of course, you leave out the beef, because there is fat attached to beef, so I will go along with the doctor on everything except the beef.

PROBLEMS OF REHABILITATION IN PATIENTS WITH CEREBRAL PALSY*

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Rehabilitation may be defined as "the restoration of one's health and efficiency." Strictly speaking, therefore, it would apply only to those who once enjoyed full health and seek again to regain it. The term, however, has been extended to include those handicapped individuals suffering from congenital and post-natal afflictions which require development and training toward a more normal response level.

Plans for rehabilitation and training not only depend on the degree and extent of the physical handicap, but to a large degree upon the mental qualities that remain and are capable of social and educational adaptation.

The first step is one of classification of the patient. The clinition will have little difficulty in separating the cerebral palsy patient from the polio case. It is not, however, always an easy matter to separate the "cerebral palsy" patient from the "high spinal spastic." The former may have much mental difficulty, with spastic diplegia—the latter great spasticity, but an active mind.

As the symptoms of cerebral palsy vary from mild weakness and an increase in reflex activity and muscle tone to severe paralysis with contraction and contracture, the diagnostic difficulties are numerous.

Cerebral palsy is more difficult to define because the term has come to include almost all types of congenital and acquired dysfunction of the motor system that can be ascribed to the brain. Although it is generally applied to the infant or child, the same manifestations of paralysis and spasticity arise in the adult after certain well known vascular lesions.

Many cerebral palsy clinics have sprung up over the country to cope with the problems which the spastic, the athetoid, and the bulbo-ataxic patients present. It is estimated that approximately 22,000 of these individuals live in the state of Pennsylvania, and that there are more than 300,000 scattered throughout the United States.

The most familiar instance of this condition is the spastic child with scissor gait, known as Little's Disease. Here the legs and feet are usually more involved than the upper extremities and the child tends to walk on the toes, crossing the legs due to adductor spasm. The hands and arms are often affected and spastic, so that they are drawn tightly up against the chest. This type may or may not manifest delayed or defective speech. As this is a cortical type of involvement, usually at the vertex, there may be other brain areas involved so that mental retardation and defective development are common.

The next readily recognizable type is the spastic athetoid. Here the motor pathways have been involved, perhaps in the mid-brain at the striatal level, so that great disturbance of co-ordination exists and the patient has difficulty in movement because of altered tone as well as power. These are the types that twist and squirm in trying to perform simple acts. Speech may be explosive and drooling is common.

The patient's plight is so altered from the normal manifestations of movement or even paralysis, that he becomes an object of pity rather than close study. The appearance of drooling has been associated with childish idiots and imbeciles, so that this group suffer not only from the physical handicaps, but also from social isolation. Most of these patients have fairly good understanding and intelligence, and thus suffer more from an unjust and misunderstood mode of treatment offered to them.

The athetoid types usually have excellent memory and musical possibilities. We have rehabilitated those who have speech and intellectual defects and cannot enter conventional school courses because of their physically grotesque appearance by teaching them music such as organ work and trap drums.

I have two young men who have learned to play the organ well enough to take over the duties in church, where they are well cared for and understood by the parishioners and have given great satisfaction to their parish. Another has done "solo" work on the trap drums in a night club, receiving, I am told, $12.00 a night. His family take him to the club and call for him in a taxi. The net earnings of this otherwise dependent member of the family exceeds a brother who has a good

* Read before the Medical Society of Delaware, Wilmington, October 9, 1945.
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position in a shipyard. The boy's athetoid movements are partly screened by palms and some of the "antics" are enjoyed by the patrons as being high class jazz performance.

Another child with extensive frontal lobe involvement I have carried through nine years of musical implantations in order to get speech and certain necessary activities that his original mental level could not anticipate. The boy is now able to compose, transpose, and enlarge on any musical theme he hears. He cannot read and scarcely write. The spastic paralysis of the hands makes conventional piano music impossible, but somehow the fingers of both hands combine to bring forth rich and fine music in new and pleasing combinations. Without this accomplishment, the child would be thought to be a spastic idiot. Rehabilitation of this type requires careful and long term planning. There is much work and thought needed in this field of medicine today.

The mixed spastic ataxic type that occurs with lesions of the brain at the level of the bulb and cerebellum is best exemplified by the symptoms of Friedreich's ataxia. Here, like the first group mentioned above, the cerebral diplegias, correction and retraining has been left to the orthopedic surgeon, the physiotherapist, and occasionally some suggestion or help has been added from the neurological point of view.

Perhaps the most important group of all and the one that offers the most promise of mental and physical rehabilitation is what we have chosen to call the *high spinal spastic*. These patients have invariably been included in the cerebral palsy group because of the spastic arms and legs simulating the cortical type. They are, however, quite distinct and are due for the most part to injury, pressure, or inflammation of the cervical cord and medulla at the level of the juncture between the skull and atlas. Many birth injuries occur at this level and are not detected. The head is twisted off the spinal column in the mechanism of pulling and rotating the infants head at birth. Forceps traction may be very harmful in this respect. More and more neuropathological lesions with high sounding names are appearing under various diagnoses where careful x-ray and neurological examination show the lesion to be an anterior displacement of the atlas, axis or third cervical vertebra. The resulting pressure on the motor fibers at the decussation of the pyramids which occurs at this level along with local pressure of the odontoid may and does give paralysis of the lower extremities with marked spasticity which also includes the arms and hands.

As the brain is not at fault and is uninjured, this group offers excellent possibilities of mental progress and the chief problem concerns management and modification of the spasticity and contracture. Great improvement has been observed in several cases following decompression of the posterior ring of the atlas and axis. This permits additional space and improved circulation to the spinal cord and medulla and thus eventually assists in subsequent correction of the orthopedic problems.

In one instance, I operated a four days infant with immediate and complete relief of the spastic paralysis. The child is three years of age at the present time, and progressing without other correction for the spastic paralysis that originally involved both arms and legs. Recently, a ten months baby was decompressed over the high cervical cord for marked spastic paralysis of the lower extremities. The ring of the atlas, the axis, third and fourth laminae were removed. The spasticity and paralysis have so abated that crawling movements are now spontaneously being attempted.

I call your attention to these results because of the need to carefully distinguish the high spinal spastic from the cerebral palsy type, as such correctional measures are not possible in the latter but require all the skill of the orthopedic surgeon, correctional exercises, and supports.

Time does not permit discussion of the methods of vocational rehabilitation now in use in the hospital and armed forces. They are well known to all and each has its distinct advantage and purpose.

I would in closing, like to call your attention to what may prove to be a more promising attack on the spastic paralysis encountered in the cerebral palsy group from the standpoint of rehabilitation of movement than the measures we have depended upon in the past. The results have been welcomed by the orthopedic

surgeon and physio-therapist alike and are worthy of an honest trial and effort.

In the cerebral palsy group, the injury lies chiefly on the cortex, or in the mid-brain. Many simple patterns of movement arise at the level of the spinal cord and medulla. It has been possible to train cerebral spastics along the lines of simple amphibian patterns of movement, such as swimming and crawling movements, when apparently paralyzed to the higher patterns of the cortex.

Co-ordination in the athetoid group has become much easier and correction of many defects of posture and progress in the spastic has been accomplished by reverting to patterns of movement common to the infant rather than attempting skilled activities more acceptable to man's concept of a normal adult.

The physio-therapy department of the Neuro-Physical Rehabilitation Clinic has developed some of these primitive patterns. Details of treatment are available upon request. We have found the old adage that "one must creep before he walks" to apply to this group. Certain free swimming movements in our experience have led directly to better ultimate progress of the child in unsupported efforts at balance and in walking.

The problems of rehabilitation vary with each individual and each case becomes a special need for study. As a group, the cerebral palsies present a spectrum from the mildest types without need for physical correction to the spastic quadraplegia with mental defects beyond hope of rehabilitation.

Progress is gradually being made in the classification and adaptation of activity to the general needs of the patient and the community.

The greatest need today is a better understanding of this group and an effort to widen the social circle of those whose mentality is good, though the physical handicap may be severe.

250 *So. 17th St.*

DISCUSSION

DR. GEORGE J. BOINES (Wilmington): Dr. Fay has given us valuable suggestions for the rehabilitation of a group of patients who have been heretofore considered hopeless. However, we have had little work so far in the actual prevention of the deformities which render a spastic almost a useless individual in many cases. If one checks up in the textbooks and literature as to the actual treatment of spastics one finds the same thing as for poliomyelitis patients, that is muscle transplantation, corrective exercises, and other surgery, but very little has been done for the actual prevention of the deformities themselves.

In the past three or four years great emphasis has been placed on the treatment of deformed patients through physical medicine. This has been done chiefly through work of the Baruch Foundation and armed services in rehabilitating deformed individuals.

We know that through the use of psychotherapy, occupational therapy, muscle rehabilitation and prostigmin in the acute, subacute and chronic stages of poliomyelitis, we have been able to correct deformities that were not being corrected by surgical methods.

We have tried the same methods with a number of spastics. We have one spastic 18 years of age who has shown considerable improvement with prostigmin. We find that many of the tight and short muscles in these patients can be lengthened and the patients can be taught, through coordination exercises, to use their limbs in a better fashion than they were able to do before.

I would like to ask Dr. Fay if he does not think that this opens an important field in the treatment of spastics, that is, getting the patient early and making early diagnosis and trying to prevent the deformities which follow, by the means outlined above.

One thing, in Delaware we have no such thing as a Spastic Clinic. Spastics, at home, become a burden to their family and to society, and receive little treatment. The only treatment I know of that they receive is an occasional or yearly visit by some man prominent in spastics, and are then sent home, as nothing can be done.

I am sure through the suggestion of Dr. Fay a lot can be done for these cases, as has been done for chronic poliomyelitis patients.

DR. M. A. TARUMIANZ (Farnhurst): I would like to ask a question. But first, I would also like to emphasize that I enjoyed Dr. Fay's presentation. I have heard so much about Dr. Fay's present attempt to find more physical methods to correct the unfortunate situation of cerebral palsy cases, or spastics, or in several spastic cases. How-

ever, Dr. Fay spoke of those who still have sufficient mentality to become useful citizens in some way, citing many cases who apparently have still ability to become musicians, and so on. But is it not true that one cannot establish the level of intellectual endowment and the remaining intellectual capacity unless one has a proper approach by means of psychological tests? Is it not true that we should attempt to classify these youngsters in two groups—imbeciles, or morons; and fairly well established mentality?

So I would like to ask whether in his cases he has exercised that approach of psychological tests and what his findings have been?

I enjoyed Dr. Fay's paper very much.

DR. FAY: I believe the question, of course, of early diagnosis is of prime importance as in almost every disease, and correction is often best, perhaps, by not only early diagnosis but by immediate physical measures that are indicated for that individual. However, we find that each case is a law unto itself; it must be analyzed and one cannot just provide a general treatment for cerebral spastics. I think it is as wide as the spectrum of any other material that we become associated with. So that the first need is one of careful classification. If we choose to accept the intellectual levels current today one, however, may be easily misled in the guiding of these people, because, unfortunately, the mental and intellectual tests today are not standardized as far as the function of the brain is concerned, and one person may in certain situations meet a higher level than on other occasions. We believe that might have something to do with the amount of food and fluid intake, and that a patient should be as carefully attended on the feeding program as when you take basal metabolism. However, that has not come into vogue yet. We have in our clinic Dr. Lew Kennedy, the author of "Rehabilitation of Speech" and she is giving us the mental levels, and I am trying to coordinate the findings of others with some of the physical factors. Now we are enjoying the confusion encountered by loading our children with food and fluid on certain mornings and finding them falling down on tests. On the other hand we will dehydrate and balance this carefully to get better fluid supply to the brain and they will come in with different reports of this child getting better. So I feel we need mental tests, but standardized mental tests — we can't be satisfied with the Binet tests and the rest of the tests today, any more than we can if we were to take a basal metabolism of everybody in this room, irrespective of what he had for breakfast today.

Therefore, I feel that drawing conclusions may be clarified enough to give us a pattern that is consistent, but until the mental testers can become more organized and consistent on a standardized basis I am waiting and watching and correlating what I can. But I am far from being able to drawing conclusions and perhaps those will not be forthcoming for a good many years.

HEALED DISSECTING ANEURYSM OF THE AORTA, WITH SIGNS OF AORTIC INSUFFICIENCY

Case Report

J. RICHARD DURHAM, M. D.,*
Wilmington, Del.

Among the many cases of dissecting aortic aneurysm reported there are several which have presented the symptoms and signs of aortic regurgitation, resulting in an antemortem diagnosis of aortic valvular disease. A review of the literature by Gouley and Anderson[1] showed in such cases that the dissection proceeded downward to the level, or nearly so, of the aortic ring. They believed that the simulation of aortic valvular disease was dependent on the proximity of the aneurysm to the valve ring and not on the extent of the dissection. It is our purpose to present such a case, to discuss its mechanism, and to illustrate an interesting example of digitalis intoxication.

C. W., a 68-year-old white male, was admitted to the St. Francis Hospital on April 19, 1941. He was of Polish extraction but had lived in this country for the last 20 years. Very little history could be obtained because of his inability to speak English; however, the following facts were elicited: patient had been taking "medicine" at home for three weeks because of shortness of breath, cough, and swelling of the ankles. He had appar-

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ently always enjoyed good health except for an attack of "rheumatism" at the age of 35. There was no history of chancre. Five years prior to the present illness, patient remembered experiencing sudden severe substernal pain while chopping wood. This was of short duration, did not recur, and no treatment was received, What other history could be obtained was of no significance.

Examination revealed an orthopneic, emaciated white male, with visible cervical pulsations related to systole. There was moderate pitting edema of both ankles, some cyanosis of face and nail beds, and a Quincke's pulse. Blood pressure in the right arm was 222/52 and in the left arm 220/0. The radial pulse was of the Corrigan type and the rate varied between 36 and 40 beats per minute. The chest was emphasematous with dullness and moist rales at both bases. A diffuse thoracic shudder occurred with each systole and the apex beat was palpable in the 6th interspace in the anterior axillary line. A loud diastolic murmur was heard in the aortic area and transmitted downward over the precordium, and a presystolic murmur was heard at the apex. Percussion revealed the heart markedly enlarged to the left with no evidence of broadening at the base. Examination of the abdomen revealed a palpable liver edge which was smooth and not tender. Duroziez's murmur was present over the femoral vessels, and the systolic blood pressure of the lower extremities was slightly higher than that of the upper extremities. The remainder of the physical examination was non-contributory. The provisional diagnosis was aortic insufficiency, rheumatic; complete heart block, and possible digitalis intoxication.

Blood chemistry revealed the following: blood urea 55 mg%, creatinine 2 mg%, Kolmer and Kahn, negative. Blood count showed moderate secondary anemia and urinalysis was negative except for a trace of albumin.

Because of the possibility that the patient had been treated with digitalis before admission and in order to determine the nature of the heart block an electrocardiogram (Fig. 1) was taken on the day of admission. This showed a ventricular rate of 40, and marked ST-T changes in leads I, II & IV F suggesting digitalis intoxication. The ST configuration in lead IV F is quite unusual. Digitalis was withheld and the patient treated with oxygen, salyrgan, atropine and adrenalin in an at-

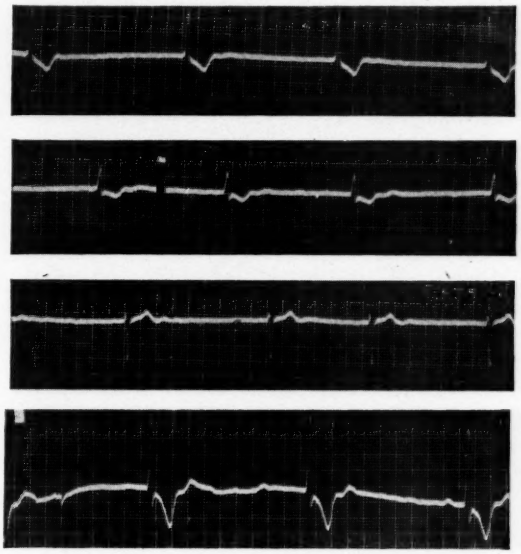

Fig. 1. EKG taken 4/19/41. Auricular fibrillation with idioventricular rhythm, left axis deviation, and ventricular rate of 40. Note St configuration in lead IV F.

tempt to overcome the heart block and restore cardiac compensation. The pulse gradually increased over a period of two weeks, becoming more irregular. A second electrocardiogram (Fig. 2), taken on May 6, 1941, showed a ven-

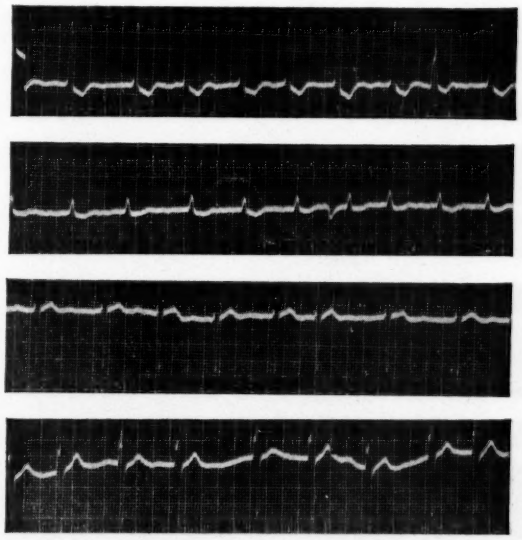

Fig. 2. EKG taken 5/6/41. Auricular fibrillation with disappearance of heart block. Ventricular rate 84-92. Note change in ST configration in leads II and IV F. Evidence of left ventricular strain persists.

tricular rate of 92 and a disappearance of the idioventricular rhythm. The changes in the ST-T segments, most notable in lead IV F, occurred following withdrawal of digitalis. This case again illustrates the need for caution when administering digitalis to older cardiac patients.

Little improvement followed the increase in heart rate, and the patient expired suddenly on the 24th day of hospitalization.

Autopsy performed by the pathologist, Dr. Douglas M. Gay, on the day of death revealed (in pertinent part) a greatly hypertrophied heart measuring 17.5 cm. in transverse diameter and weighing 700 grams. The myocardium was homogeneous with no evidence of scarring. All valves appeared competent, with no trace of sclerotic changes, and the aortic ring was not dilated. Eight-tenths (.8) cm. above the aortic valve ring there was discovered a transverse split in the aorta 6 cm. long (Fig. 3). The central portion of this lesion opened into a rounded bulge of the outer wall of the aorta, measuring 2 cm. wide and 2 cm. deep. The split involved the intima and this layer of the aorta formed a valve like semilumar flap which projected into the lumen of the vessel. The inner layer of the lesion consisted of pale grey tissue, and there were several strands of scar tissue in the lateral edges of the split. The aorta showed no gross evidence of arteriosclerosis, and retained a normal amount of elasticity. The coronary vessels were not diseased. The liver revealed evidence of passive congestion.

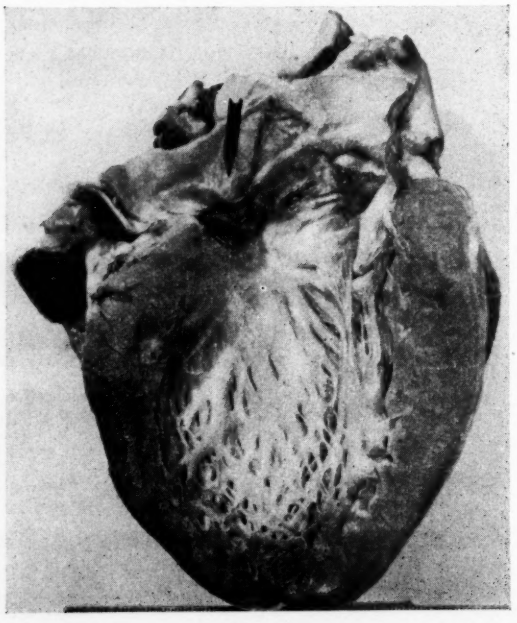

Fig. 3. Section of heart showing left ventricle and first portion of aorta. The free edges of the aortic valves and lip of the aneurysmal sac have been stained with methylene blue. Note myocardial hypertrophy.

DISCUSSION

The mechanism of regurgitation in this case was caused by the lip of the aneurysm interfering with closure of the aortic valves. One can visualize from figure 3 that the blood column in the aorta following systole would easily project the lip of the sac into the lumen of the valve ring. It can be reasoned that the regurgitation was complete since it has been previously shown in cases of dilated aortic rings, in which central leakage of blood occurs in diastole, that eroding and roughening of the free margins of the leaflets resulted. No such changes were found in this case. Larger aneurysmal sacs with more extensive dissection may produce a similar clinical picture through dilatation and distortion of the aorta[2], and still larger dissection may produce symptoms of insufficiency entirely from the leakage of blood into the sac[3]. In this case it is surprising that the aorta showed no degenerative or inflammatory lesions at autopsy, other than the sac described. It is possible, however, that dynamic aortic dilatation may have been a factor in addition to the mechanical interference with valve function described above. Unfortunately, no x-ray studies of the heart were made. The onset of the intimal tear may have occurred five years prior to the present illness at which time the patient was seized with severe chest pain while chopping wood. Eskelund[2] believes rupture occurs in an area predisposed by degenerative changes when the blood pressure rises beyond the limits of vascular resistance or following a relatively unnoticed trauma.

Aortic regurgitation is not uncommonly associated with a presystolic murmur, and such a murmur was heard in this case. The most generally accepted explanation is that it is due to the anterior leaflet of the mitral valve being pushed into the path of the blood stream entering from the left auricle by the regurgi-

tation through the aortic orifice. Gouley[8] believes the Flint murmur can be produced by a characteristic deformity of the right leaflet of the aortic valve which he has described but which was not present in this case.

SUMMARY

A case of healed dissecting aneurysm simulating aortic insufficiency is presented with a discussion of the mechanism involved. This etiologic possibility should be considered in aortic insufficiency of non-luetic and non-rheumatic origin.

623 *Delaware Avenue.*

BIBLIOGRAPHY

1. Gouley, B. A., and Anderson, E.: Chronic Dissecting Aneurysm, Ann. Int. Med., 14: 978-990 (Dec.) 1940.
2. Eskelund, V.: Aortic Insufficiency as Result of Spontaneous Rupture of Aorta, Ugeskrift for Laeger, 103: 227-256, 1941.
3. Resnik, W. H., and Keefer, C. S.: Dissecting Aneurysm with Signs of Aortic Insufficiency, J. A. M. A., 85: 422, 1925.
4. Reisinger, J. A.: Dissecting Aneurysm, Arch. Int. Med., 65: 1097-1115 (June) 1940.
5. Rattino, A.: Medial Degeneration Cystic Variety in Unruptured Aortas, Am. J. Heart Dis., March, 1940.
6. Roberts, J. T.: Medionecrosis Aortae Idiopathica Cytica, Am. J. Heart Dis., 18-2: 188-200 (Aug.) 1939.
7. Finkelstein, R., and Jacobi, M.: Dissecting Aneurysm with Case Report, Ann. Int. Med., 13: 1991-1998 (April) 1940.
8. Best, C. H., and Taylor, N. B.: Physiological Basis of Medical Practice,296-298, 1939.

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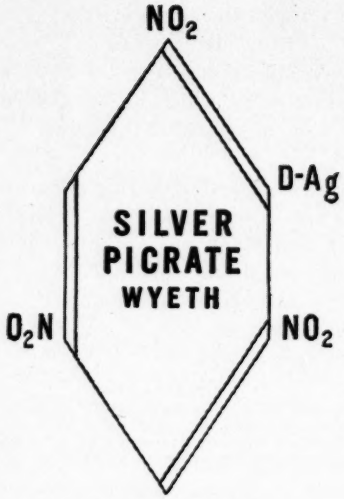

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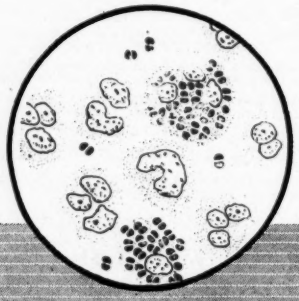

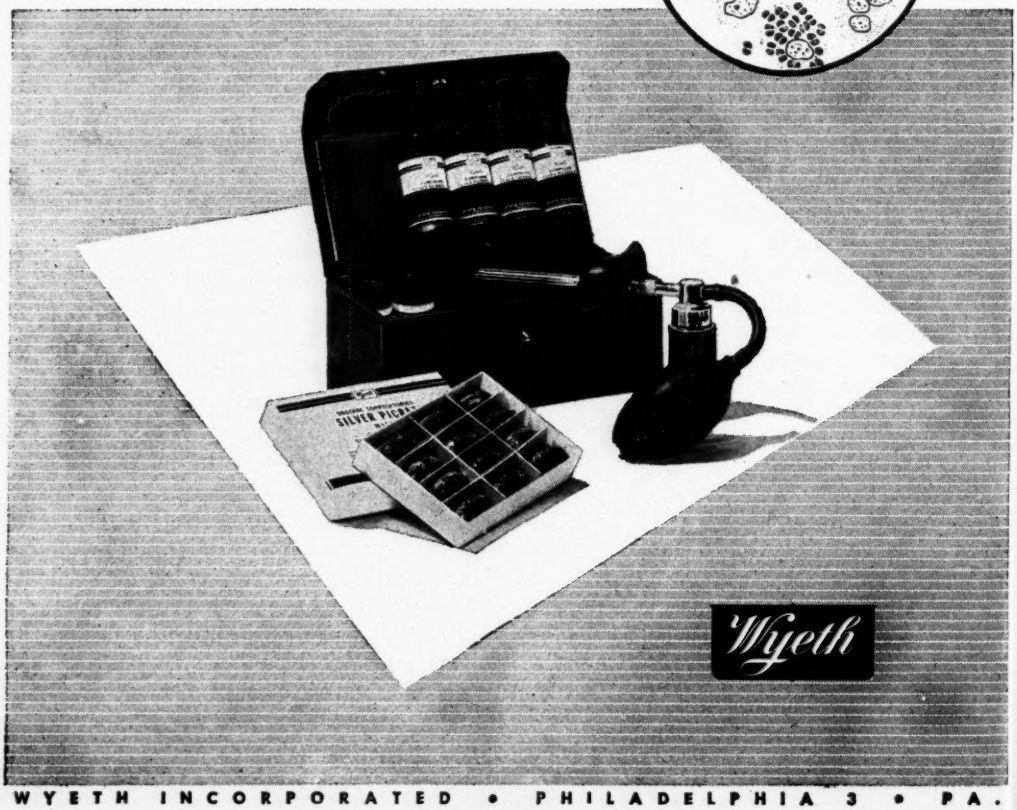

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SPECIAL SESSION

The President and Council of the Medical Society of Delaware called a Special Session of the Society which was held at the Delaware Academy of Medicine, Wilmington, on Tuesday, April 2nd, at 8:30 p. m. The importance of this Session cannot be overestimated.

The purpose of this unique session was to instruct all the members concerning the pending legislation which, if enacted into law, would revolutionize the practice of medicine in this country. Special emphasis will be placed on the Wagner-Murray-Dingle Bill which proposes, despite the quibbling of its proponents, nothing less than the socialization of medicine in the United States in a pattern closely resembling that proposed in Great Britain. Our British confreres are preparing an all-out campaign against the proposals there; we can do no less here.

The meeting was addressed by Dr. Frank Borzeu, of Philadelphia, a member of the National Physician's Committee, who is thoroughly conversant with this Bill in its entirety, and explained its details and why the medical profession is opposed to them. Doctors, who are instinctively opposed to political medicine, make poor arguments for their viewpoint unless they have had special instruction. With the facts well in hand, any physician can convince his own public of the merits of his argument, and since only an enlightened and militant public opinion can defeat the present intent of certain political groups, the unmistakable duty of each and every doctor who belongs to the Society was to be on hand. Despite the inclement weather, the attendance was gratifyingly large.

Pending the new Directory of Medical Specialists, which will not appear for several months, it is very desirous that this office have a complete list of all the diplomates in Delaware of the various American Boards for the specialties. Kindly send to the Editor your name and address, the name of the particular board, and the date your certificate was issued. Our thanks.

Also, please send us any corrections that should be made to the Directory page, published in the January issue of THE JOURNAL. Since this page cannot be published every month, it is important that any changes due to new elections, death, removal, etc., be reported promptly. Again, our thanks.

NEW NATIONAL FORMULARY

Many persons are confused by the existence of two official books of drug standards in the United States—the U. S. Pharmacopoeia and the National Formulary.

"The United States Pharmacopoeia," said Dr. Justin L. Powers, Chairman, Committee on National Formulary, American Pharmaceutical Association, "from the very beginning has restricted its scope to drugs selected by the medical members of the Revision Committee and believed by them to possess greatest therapeutic merit. This selectivity has prevented it from recognizing a large number of drugs which, judging by the extent of actual use by the medical and pharmaceutical professions, must possess some merit. Briefly, admissions to the Pharmacopoeia are on the basis of therapeutic merit while admissions to the National Formulary are on the basis of extent of use. For instance, in the course of a revision of the Pharmacopoeia, numerous drugs are deleted, many of which continue to be used extensively for varying periods of time thereafter. Since the deletion does not mean that the drug immediately becomes obsolete, it is often desirable and even essential to continue to provide official standards for it."

The first edition of the National Formulary appeared in 1888. Official recognition and endorsement came when the Federal Food & Drug Act of 1906 adopted the National Formulary and the U. S. Pharmacopoeia as legal standards. Increased responsibility brought an ever increasing amount of attention to the careful preparation of the Formulary, and during the past several years its revision has been on a continuous basis with the efforts of full time employees directed solely to this work.

Public health has developed to the point where it has become apparent that many diseases cannot be controlled without full citizen understanding and participation. The provision of x-ray and diagnostic facilities, sanatoria, and rehabilitation services will not reduce tuberculosis unless people know the value of an x-ray of the chest, have one taken periodically, and take the necessary treatment if a diagnosis of tuberculosis is made. Mayhew Derryberry, Ph. D. Pub. Health Rep., Nov. 23, 1945.

BOOK REVIEW

Harofe Haivri. Vol. II, 1945, of Harofe Haivri (The Hebrew Medical Journal), a semi-annual bilingual publication edited by Dr. Moses Einhorn, has appeared.

The contents of this journal are not confined to technical medical topics, but are divided into several sections covering a variety of related subjects, such as medicine in the Bible and Talmud, old Hebrew medical manuscripts, Palestine and Health, etc. In the medical section, the following subjects are discussed: The Treatment of Heart Failure by Harry Gold, M. D., The Anemias and Their Treatment in the Light of Recent Advances by Gershon Ginzburg, M. D., and The Child and His Mental Health by Samuel J. Lipnitszsky, M. D.

Under the topic of Historical Medicine Prof. Nahum Slushtz, of Palestine, writes an interesting article on Isaac Ben A'mran, famous physician and philosopher, who lived in Keiruhan in the middle of the 18th century and served as court physician to Emir of Zaduth Alla.

In the section on Talmud and Medicine, Dr. M. Ben-Ami writes explanatory remarks on the medicine in the Bible; in addition, Dr. Z. Muntner discusses the social status of the physician in the Talmud.

Dr. Solomon R. Kagan offers an article of particular interest on the contribution of the pioneer physicians to the growth and development of the Zionist movement throughout the world. Since the Middle Ages the Jewish physician has exerted great influence on the communal life of Israel. They were not only the healers of the body and mind, but also leaders, statesmen and diplomats who fought for the rights and freedom of their brethren. The current issue discusses the contribution of the Russian and Palestinian physicians to the Zionist cause.

The book review section contains detailed reviews by Dr. A. Goldstein and Dr. L. Herbert of Dr. A. Sadovsky's book "A Textbook on Obstetrics," in Hebrew, published by the Hadassah Medical Organization.

There are complete English summaries of all the original articles.